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Air Pollution Assessment of Vinylidene Chloride

MITRE Corp, McLean, Va METREK Div

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AIR POLLUTION ASSESSMENT OF VINYLIDENE CHLORIDE

U.S. ENVIRONMENTAL PROTECTION AGENCY
Office of Air and Waste Management
Office of Air Quality Planning and Standards
Research Triangle Park, North Carolina 27711

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Vinylidene chloride is toxic to laboratory animals and can be fatal at sufficiently high dose levels. Liver is the prime target organ of vinylidene chloride in mammals. Hepatic injury can occur rapidly after inhalation exposure. Chronic exposure to low levels of vinylidene chloride can result in liver and kidney damage. Vinylidene chloride is a central nervous system depressant reported to have a narcotic effect. Mutagenicity of vinylidene chloride in micro-organisms indicates a need for investigation of its carcinogenicity.

The population at risk due to vinylidene chloride exposure is composed primarily of workers in industrial or commercial operations manufacturing or using it. Airborne emissions of vinylidene chloride are not likely to pose a significant risk to the general population. Emissions during production, storage, and transport can be controlled by methods similar to those planned for control of vinyl chloride.

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by

J. Hushon and M. Kornreich

METREK Division of The MITRE Corporation
1820 Dolley Madison Boulevard
McLean, Virginia 22101

Contract No. 68-02-1495

EPA Project Officers: Richard Johnson and Joseph Cirvello

Strategies and Air Standards Division

Prepared for

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ABSTRACT

Vinylidene chloride is toxic to laboratory animals and can be fatal at sufficiently high dose levels. Liver is the prime target organ of vinylidene chloride in mammals. Hepatic injury can occur rapidly after inhalation exposure. Chronic exposure to low levels of vinylidene chloride can result in liver and kidney damage. Vinylidene chloride is a central nervous system depressant reported to have a narcotic effect. Mutagenicity of vinylidene chloride in microorganisms indicates a need for investigation of its carcinogenicity.

The population at risk due to vinylidene chloride exposure is composed primarily of workers in industrial or commercial operations manufacturing or using it. Airborne emissions of vinylidene chloride are not likely to pose a significant risk to the general population. Emissions during production, storage, and transport can be controlled by methods similar to those planned for control of vinyl chloride.

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I. SUMMARY

Vinylidene chloride, also known as 1,1-dichloroethene or 1,1-dichloroethylene, is used in combination with other monomers to produce copolymers with such desirable properties as high strength, thermal stability, impermeability and resistance to attack by solvents, fats and oils. Saran, formerly a Dow Chemical Corporation trademark, is now used in the United States as a generic term for high vinylidene chloride content polymers. Vinylidene chloride copolymers are extruded as films and fibers. Resins made from vinylidene chloride copolymers are used as binders and coatings. Vinylidene chloride monomer is used in production of two modacrylic fibers. Vinylidene chloride is also used as an intermediate in the manufacture of 1,1,1-trichloroethane.

Vinylidene chloride is a clear liquid at room temperature. It is insoluble in water, but soluble in most other polar and nonpolar solvents. Vinylidene chloride is a very volatile compound having a boiling point, at atmospheric pressure, of 31.56°C. It is flammable at vapor concentrations between 7 and 16 percent by volume in air. In the presence of air or oxygen, liquid vinylidene chloride forms a highly explosive peroxide compound at temperatures as low as -40°C. Any dry composition containing more than 15 percent peroxide detonates from a slight mechanical shock or heat. For this reason, a peroxide formation inhibitor is usually added to vinylidene chloride prior to storage. Environmental reactions of vinylidene chloride with oxygen

or ozone in the presence of light can result in formation of formic acid, carbon monoxide, formaldehyde, phosgene chloroacetyl chloride and hydrochloric acid, as well as explosive peroxides.

Vinylidene chloride is toxic to laboratory animals and can be fatal at sufficiently high dose levels through inhalation or oral exposure. The 24-hour oral LD_{50}* for a single dose of vinylidene chloride in rats is approximately 1,550 mg per kg body weight. The LC_{50}** for inhalation exposure of rats to vinylidene chloride has been reported as 15,000 ppm for deaths within 24 hours after exposure and 6,350 ppm for deaths within 14 days after exposure. The susceptibility of animals to vinylidene chloride poisoning is increased during fasting or at night.

The acute toxicity of vinylidene chloride is similar to that for carbon tetrachloride and significantly greater than the acute toxicity of vinyl chloride. A threshold limit of 10 ppm is recommended by the American Conference of Governmental Industrial Hygienists for prevention of liver or kidney damage in humans from acute exposure to vinylidene chloride. No OSHA standard has been established.

Acute inhalation exposure to vinylidene chloride would be expected to result primarily in central nervous system depression manifesting itself as narcosis, symptoms of drunkenness, or loss of consciousness. These symptoms would occur rapidly at concentrations of approximately

*LD_{50}: Mean lethal dose.
**LC_{50}: Mean lethal concentration.

4,000 ppm and should be reversible after brief exposures. Liquid vinylidene chloride is irritating to skin and eyes.

Human exposure to vinylidene chloride occurs mainly through inhalation. Animal studies have shown that toxic symptoms can result from chronic exposures to low levels of vinylidene chloride. Such exposures would be expected to result primarily in liver and kidney damage. Even when the concentrations of vapor are too low to cause an observable anesthetic effect, they may produce injury to liver or kidney.

The liver is the prime target organ of vinylidene chloride in most laboratory mammals and, apparently, in humans. Hepatic injury (hemorrhage and necrosis) occurs quite rapidly after inhalation exposure (2 to 4 hours of exposure to 2,000 ppm in fasted rats). Single oral doses of 400 mg per kg body weight were hepatotoxic to rats, causing morphological changes in liver cells and large quantities of neutral fat within the liver. The total amount of hepatic fat increased as dosage of vinylidene chloride increased. At 800 mg/kg body weight, the amount of lipid present was almost double that in controls.

The most sensitive indicators of vinylidene chloride injury to liver are disruptions of normal microsomal enzyme activity. Greater than tenfold increases in liver alkaline phosphatase activity and liver tyrosine transaminase activity have been observed in male rats 20 hours after administration of single 500 mg/kg body weight doses

of vinylidene chloride. Fasted rats are more susceptible to hepatotoxicity of vinylidene chloride than fed rats, and male rats are more susceptible than female rats.

Kidney damage resulting from vinylidene chloride exposure has been observed in experimental animals. Vinylidene chloride is a central nervous system depressant reported to have a narcotic effect on mice, cats, rabbits and guinea pigs. Lung damage has occasionally been observed during prolonged inhalation exposures.

Vinylidene chloride has been shown to be mutagenic in microbial systems mediated by microsomal enzymes from various organs. Mouse liver, kidney and lung fractions converted vinylidene chloride into mutagenic metabolites in vitro. Since most known carcinogens which have been tested in such assay systems have been found to be mutagenic, results of these tests are currently used to select environmental chemicals for carcinogenesis bioassay. On this basis, the mutagenicity of vinylidene chloride in microorganisms indicates a need for investigating its carcinogenicity. Preliminary reports from Dr. Cesare Maltoni indicate that inhalation exposure to vinylidene chloride may cause kidney tumors in male mice. Vinylidene chloride is currently on test in the National Cancer Institute Bioassay Program. The National Science Foundation is also sponsoring a carcinogenesis study of vinylidene chloride.

The current domestic production of vinylidene chloride is approximately 260 million pounds per year. An industrial growth rate of 5 to 10 percent has been predicted for the next 5 years.

Vinylidene chloride monomer is produced at three locations, but vinylidene chloride may also be released during production of sarans, 1,1,1-trichloroethane, methyl chloroform, modacrylic fiber and vinylidene fluoride. Total vinylidene chloride emissions to the environment have been estimated to be between 1 and 2 million pounds per year. Due to its volatility most of the emissions will be rapidly airborne even if they originate as spills of liquid monomer. If the predicted improvements are assumed for the PPG plant at Lake Charles, Louisiana, the emissions at ground level 500 m downwind at all facilities would range from 0.16 to 0.31 mg/m^3 for a 1-hour exposure and 3.72 to 7.32 mg/m^3 for a 24-hour exposure. Except during adverse atmospheric conditions, the exposure levels of the nearby resident populations are probably not significant. The population at risk due to vinylidene chloride exposure is composed primarily of workers in industrial or commercial operations manufacturing or using vinylidene chloride.

Airborne emissions of vinylidene chloride during monomer production, storage and transport can be controlled by methods similar to those planned for control of vinyl chloride emissions, since the compounds are chemically similar and are often both present during polymerization.* These methods include proper design of vents and vapor condensing apparatus, proper location of vents within the plants,

*This is true at Dow facilities where the vinylidene chloride monomer is produced, stored and then polymerized with vinyl chloride monomer. Most polymerization facilities, however, have only vinylidene chloride present with other monomers (Strasser, 1977).

and proper storage, handling, transportation and disposal procedures. The monitoring of vinylidene chloride emissions during distribution operations is recommended.

Vinylidene chloride may be present in liquid effluent from production plants. Since vinylidene chloride has low solubility in water, it would soon vaporize. Wastewater could therefore become a source of airborne emissions. Monitoring of liquid effluent from plants manufacturing or using vinylidene chloride is recommended to determine the need for control.

II. AIR POLLUTION ASSESSMENT REPORT

A. PHYSICAL AND CHEMICAL PROPERTIES

1. Physical Properties

Vinylidene chloride is also known as 1,1-dichloroethene, 1,1-dichloroethylene, VDC, vinylidene chloride monomer, Sconatex, 1,1-DCE, and vinylidene dichloride. It is a clear liquid with a low viscosity at room temperature (Merck and Co., 1968; Weast, 1976). It possesses a pleasant, sweet odor similar to those of carbon tetrachloride and chloroform (Merck and Co., 1968; Wessling and Edwards, 1967; Dow, 1974). Most persons find that at concentrations of 1000 ppm in air, it has a mild but definite odor. Many people can detect levels of 500 ppm. Vinylidene chloride vapors containing decomposition products have a disagreeable odor and can be detected at concentrations considerably less than 500 ppm (Dow, 1974). The odor threshold is inadequate to prevent excessive exposure since the threshold limit value has been set by the American Conference of Governmental Industrial Hygienists at 10 ppm or approximately 40 mg/m^3 (American Conference of Governmental Industrial Hygienists, 1976). VDC is not known to occur in nature (SRI, 1977).

Vinylidene chloride is a very volatile compound, having a vapor pressure of 660.4 mm Hg, and hence must be handled with extreme care. Volatility is such that vapor concentrations exceeding 2 percent (20,000 ppm) may readily occur in instances of a large spill in a poorly ventilated area (Dow, 1964).

Vinylidene chloride is rated as a high fire hazard (American Mutual Life Insurance Alliance, 1966) due to its low flash point and as a moderate explosion hazard (Sax, 1975). It burns with a green-edged flame. It is flammable at vapor concentrations between 7 and 16 percent by volume in air. Vapors of the liquid monomer, once ignited, burn readily (Dow, 1964). It ignites less easily than benzene, gasoline, or common hydrocarbons, but after a short period, it burns in a similar fashion (Dow, 1974). Vinylidene chloride is practically insoluble in water (0.21 gm/l). However, it is soluble in most other polar and nonpolar solvents (Weast, 1976; Wessling and Edwards, 1967).

Physical properties of vinylidene chloride are listed in Table I (Merck and Co., 1968; Wessling and Edwards, 1967 and 1971; Dow, 1974; American Mutual Life Insurance Alliance, 1966; Irish, 1967).

2. Chemical Properties

Vinylidene chloride forms an azeotrope with 6 percent methanol. Distillation of the azeotrope, followed by extraction of the methanol with water, yields pure vinylidene chloride (Taylor and Horsley, 1942).

Polymers containing vinylidene chloride show thermal stability and impermeability, hence vinylidene chloride is used in combination with other monomers to produce copolymers with desirable properties (Wessling and Edwards, 1967). Pure vinylidene chloride polymer is

TABLE I

PHYSICAL PROPERTIES

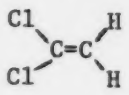
Molecular Formula:	$C_2H_2Cl_2$		
Structural Formula:			
Physical State:	Colorless liquid		
Molecular Weight:	96.95		
Freezing and Melting Point:	-122.5°C		
Boiling Point:	°C	at	Pressure (mmHg)
	31.56		760
	14.43		400
	-1.78		200
	-16.04		100
	-25.46		60
	-32.44		40
	-43.31		20
	-53.10		10
	-61.94		5
	-79.54		1

TABLE I (Continued)

Vapor Density:	3.34 (air = 1)	
Liquid Density:	Temperature (°C)	Density (gm/cc)
	-20	1.2902
	0	1.2517
	+20	1.2132
Percent in "Saturated" air:	78%	
Density of "Saturated" air:	2.8 (air = 1)	
Flash Point (Cleveland Open Cup):	5°F (4) 3°F (3)	
(Tag Closed Cup):	55°F (4) -2°F (3)	
Explosive Limits: (% by volume)	7.3 - 16.0 at 28°C	
Autoignition Temperature:	1031°F	
Latent Heat of Vaporization	6,328 ± 0.3% cal/mole at 25°C 6,257 ± 0.3% cal/mole at BP	

TABLE I (Continued)

Latent Heat of Fusion:	1,557 cal/mole
Heat of Polymerization (ΔHp):	-18.0 ± 0.9 kcal/mole at 25°C
Heat of Combustion, Liquid Monomer (ΔHc):	261.93 ± 0.3 kcal/mole
Heat of Formation (ΔHf):	
Liquid Monomer:	-6.0 ± 0.3 kcal/mole
Gaseous Monomer:	$+ .3 \pm 0.3$ kcal/mole
Heat Capacity Liquid Monomer (Cp):	26.745 cal/mole/deg at 25.15°C
Heat Capacity Ideal Gas State (Cp):	16.04 cal/mole/deg at 25.15°C
Critical Temperature (T_c):	222°C
Critical Volume (V_c)	219 cc/mole
Critical Pressure (P_c):	51.3 atmospheres
Index of Refraction (n_d^{20}):	@Temperature (°C)
	10 1.43062
	15 1.42777
	20 1.42468

TABLE I (Concluded)

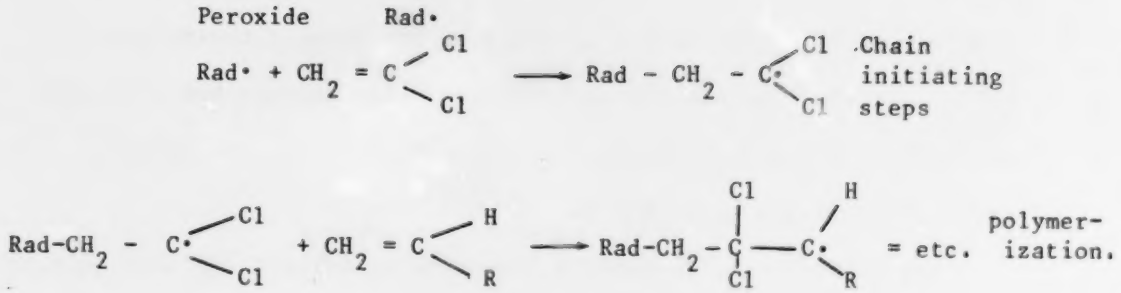
Absolute Viscosity:	Temperature (^{o}C)	Viscosity (cps)
	-20	0.4478
	0	0.3939
	+20	0.3302
Solubility of Vinylidene Chloride in Water at 25°C Wt %:	0.021	
Solubility of Water in Vinylidene Chloride Monomer at 25°C Wt %:	0.035	
Solubility Profile:	H_2O - insoluble	Acetone - soluble
	Ethanol - soluble	Benzene - soluble
	Diethyl Ether - very soluble	Chloroform - very soluble
Dielectric Constant:	4.67 at 16°C	

1 mg/liter ≡ 252 ppm and 1 ppm ≡ 3.97 mg/m^3 at 25°C, 760 mm Hg

flexible at 70°C, but at higher temperatures forms a thermoplastic solid. If the crystals are not oriented, this polymer has a tensile strength of about 8000 psi but if they are oriented, the tensile strength increases to 60,000 psi (Shreve, 1967).

The homopolymer, formerly referred to as Saran A, is difficult to fabricate and for this reason has not been extensively used. Many copolymers are possible, but only three are of commercial interest: vinylidene chloride-vinyl chloride copolymers, formerly Saran B; vinylidene chloride-alkyl acrylate copolymers, formerly Saran C; vinylidene chloride-acrylonitrile copolymers, formerly Saran F (Wessling and Edwards, 1967). In the United States, saran is now a generic term for high vinylidene chloride content polymers regardless of composition source.

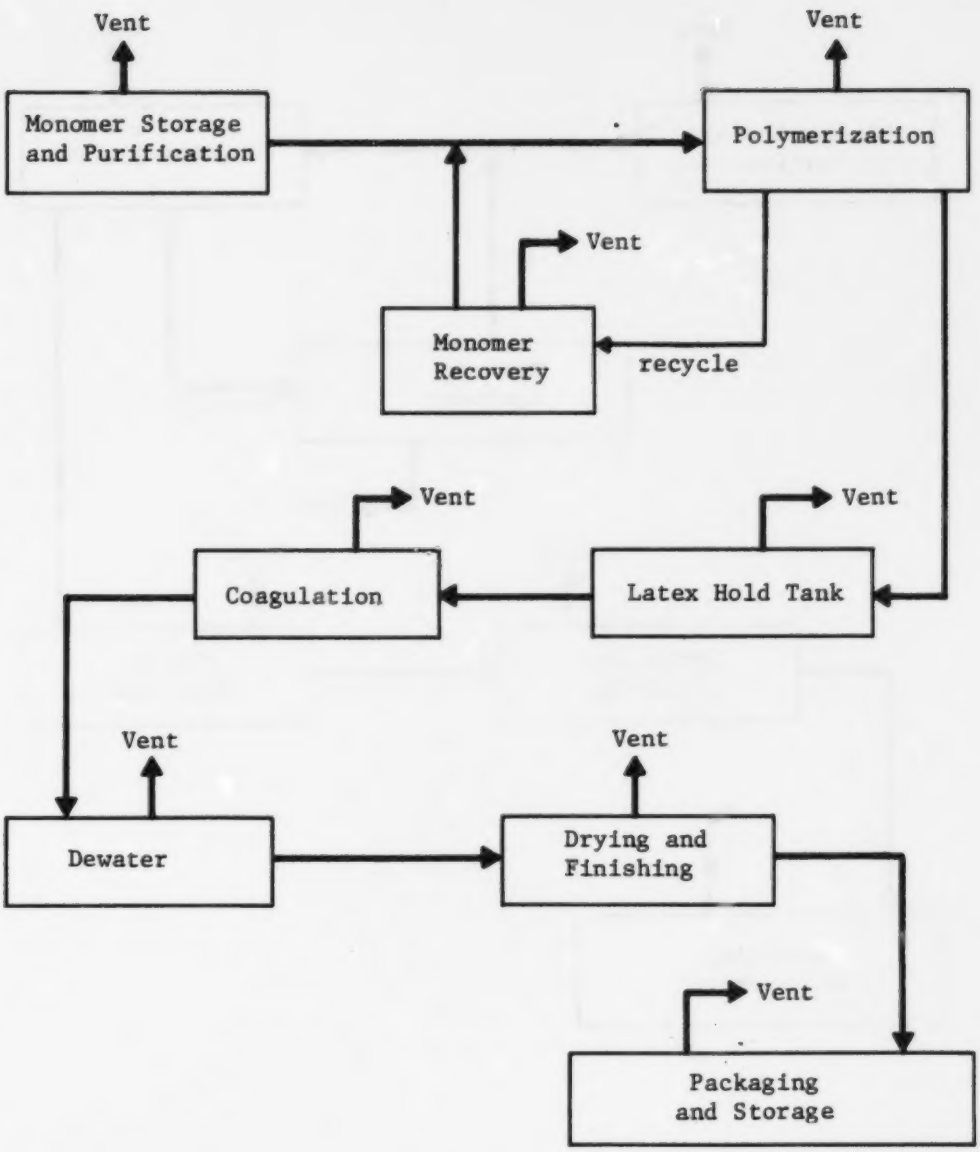
Vinylidene chloride polymerizes by both ionic and free radical reactions. However, free radical reactions predominate. When compared with other monomers, vinylidene chloride is of average reactivity. The chlorine substituents stabilize the radicals in the intermediate state of an addition reaction. Since the chlorine substituents are strongly electronegative, they polarize the double bond and make it susceptible to anionic attack. For the same reason, a carbonium ion intermediate would not be favored (Wessling and Edwards, 1967). Free radical polymerization to yield long-chain polymers requires the presence of a small amount of an initiator such as peroxide. This radical adds to a monomer molecule and generates another free radical (Rad•) (Morrison and Boyd, 1965).



The 1,1-disubstitution causes significant steric interactions in the polymer (Flory, 1953) as evident from the heat of polymerization (see Table I). If this is corrected for the heat of fusion, a value significantly less than the theoretical value of 20 Kcal/mole for the conversion of a double bond to two single bonds is attained. The steric strain does not appear to affect the addition step nor does it favor depolymerization (Wessling and Edwards, 1967).

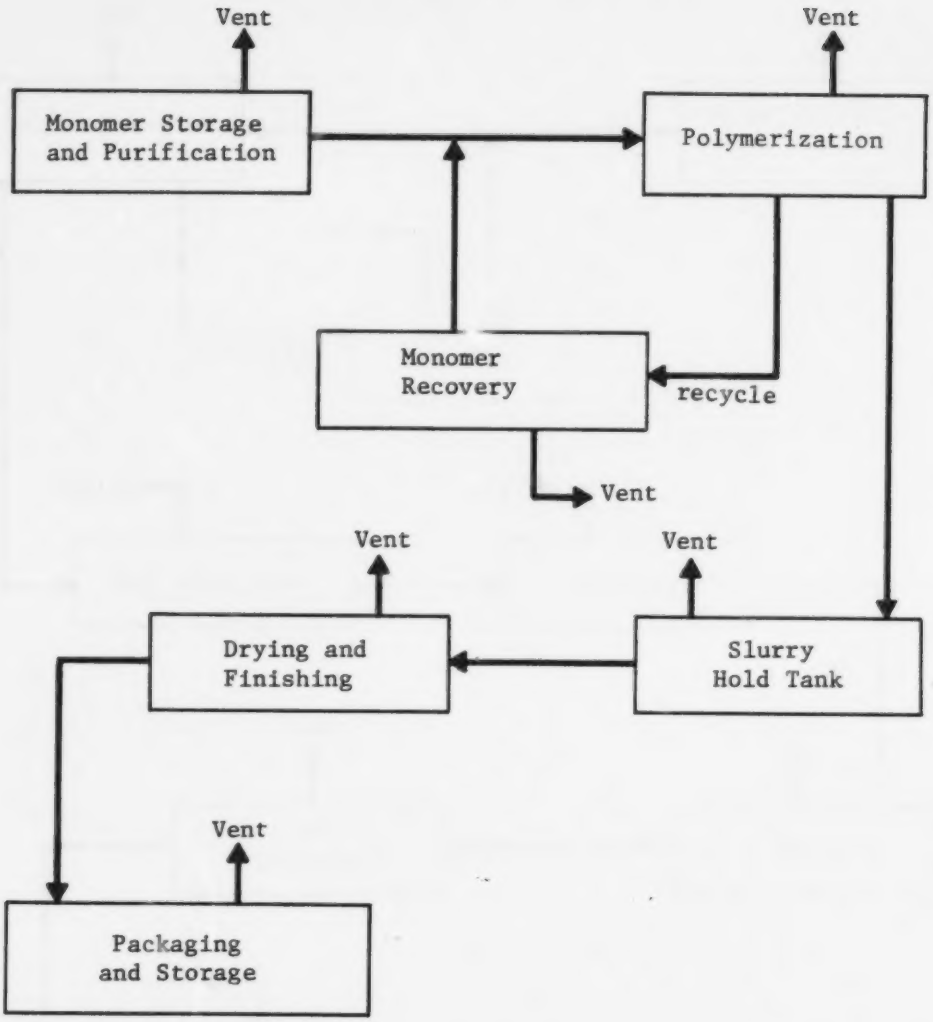
Commercial polymerization procedures are dictated to some extent by the desired polymer characteristics. Emulsion polymerization and suspension polymerization are the preferred industrial processes. The emulsion polymerization procedure is used directly to make a latex coating or the latex may be recovered in dry form. This process has the advantage over suspension polymerization of shorter reaction times (7-8 hrs at 30°C) and a high degree of control over the polymer composition. The suspension polymerization procedure, used for molding and extrusion resins, occurs more slowly but requires fewer ingredients. With suspension polymerization, improved stability and decreased water sensitivity are obtained (Wessling and Edwards, 1967). Figures 1 and 2 illustrate representative process diagrams (Strasser, 1975).

FIGURE 1
SARAN EMULSION RESINS



Source: J. P. Strasser, Dow Chemical, U.S.A., personal communication, October 21, 1975.

FIGURE 2
SARAN SUSPENSION RESINS



Source: J. P. Strasser, Dow Chemical, U.S.A., personal communication, October 21, 1975.

Lacquer resins made from vinylidene chloride copolymers are very resistant to gas and moisture transmission, and attack by solvents, fats and oils; are of high strength; and can be heat sealed. These resins are used like the extruded VDC-containing films in food packaging since they adhere to a wide variety of copolymers. They are also used as interior coatings for ship tanks, railroad tank cars, and fuel storage tanks; coatings for steel piles and structures; and binders for coatings for magnetic tapes, audiotapes, videotapes, and computer tapes (Wessling and Edwards, 1971).

Vinylidene chloride containing latexes form a water resistant coating. As a result, they are used as barrier coatings on paper products and plastic films. They are also combined with cement to form high strength mortars and concretes, and as binders for paints and woven fabrics (Wessling and Edwards, 1971).

Extruded vinylidene chloride-containing fibers are used in a variety of applications including automobile seat covers, outdoor furniture, agricultural shade cloth, and filter fabrics. Chemically resistant tubes, rods, pipes, and pipe liners are also made of extruded fibers (Wessling and Edwards, 1971).

In the United States, vinylidene chloride is used as a comonomer in the production of two modacrylic fibers, VEREL® and SEF®. An estimated 12 million lbs/yr of VDC are used to make VEREL® and 0.9 million lbs/yr for SEF® (SRI, 1976).

A typical analysis of commercial-grade vinylidene chloride monomer (excluding inhibitors) is: vinylidene chloride 99.8 wt%, trans 1,2-dichloroethylene, 900 ppm; vinyl chloride, 850 ppm; 1,1,1-trichloroethane, 150 ppm; cis 1,2-dichloroethylene, 10 ppm; and 1,1-dichloroethane, ethylene dichloride, and trichloroethylene each less than 10 ppm (Wessling and Edwards, 1971).

Inhibitors are generally added to vinylidene chloride monomer preparations shortly after production. These inhibitors prevent polymerization and preserve the quality of the product during storage and shipment. Hydroquinone mono-methyl ether (MEHQ) at 200 ppm is generally employed as an inhibitor. Phenol, p-methoxy phenol, thymol, hydroquinone, alkylamines, and organic sulfur derivatives may also be used as stabilizing agents (Hardie, 1964). MEHQ-inhibited vinylidene chloride is generally preferred for most applications since this level of MEHQ is generally overcome by polymerization initiation. To use the phenol-inhibited vinylidene chloride, however, the phenol must first be removed by alkali extraction or distillation (Wessling and Edwards, 1967; Dow, 1974).

Detailed discussions of the chemistry and properties of polymers containing vinylidene chloride can be obtained in Wessling and Edwards, 1967 and 1971; Hardie, 1964; Schildknecht, 1952; and Shelton et al., 1971.

Vinylidene chloride, in the presence of air or oxygen, without inhibitor, forms a complex peroxide and polyvinylidene chloride at temperatures as low as -40°C. The peroxides are highly explosive and any dry composition containing more than 15 percent peroxide detonates from a slight mechanical shock or heat (Wessling and Edwards, 1967). Reaction products formed with ozone are especially dangerous. The decomposition end products of vinylidene chloride peroxides are formaldehyde, phosgene, and hydrochloric acid. The presence of a sharp acrid odor thus indicates oxygen exposure and the possibility of peroxides (Dow, 1964).

The presence of peroxides can be confirmed by the liberation of iodine from a slightly acidified dilute potassium iodide solution. Any formation of insoluble polymer also indicates peroxide formation (Wessling and Edwards, 1967). Only the dry polymer is violently explosive (Dow, 1974).

Vinylidene chloride which contains peroxides may be purified by washing it several times with either 10 percent sodium hydroxide at 25°C or with a fresh 5 percent sodium bisulfite solution (Dow, 1974).

Vinylidene chloride is quite reactive toward the hydroxyl group and oxygen radical (half life of < 1 day), but not CO_2 (half life of about 3 years). Hence any releases of vinylidene chloride to the environment would not persist long prior to oxidation (Dilling et al., 1976). The resulting principal products are formic acid, carbon monoxide, chloroacetyl chloride, hydrochloric acid, phosgene and formaldehyde (Brown et al., 1975; Gay et al., 1976).

Under appropriate conditions, vinylidene chloride also reacts with hydrogen bromide (Francis and Leitch, 1957), di-sodium cysteinate (Reinhardt, 1947), sodium n-propylthiolate (McKinney et al., 1959), and hydrogen chloride (Shelton et al., 1971). Of the above reactions the only one likely to be of potential environmental significance is that with hydrogen chloride producing methyl chloroform. By far the reaction of greatest environmental significance is that of vinylidene chloride with oxygen, especially O_3, resulting in the formation of chloroacetyl chloride, hydrochloric and formic acids, phosgene and formaldehyde (Gay et al., 1976).

B. EFFECTS

1. Humans

Several toxic effects have been attributed to the inhalation of vinylidene chloride by humans. The inhalation of vinylidene chloride produces varying degrees of narcosis depending upon the amount inhaled (Wessling and Edwards, 1970). Acute exposure to high concentrations of vinylidene chloride would be expected to result primarily in central nervous system depression and an associated symptom of drunkenness which would progress to unconsciousness (Irish, 1967). These symptoms would be produced rapidly at concentrations on the order of 4,000 ppm. If the exposure is of short duration, complete recovery from this anesthetic effect may be expected (Dow, 1974).

Liquid vinylidene chloride can be irritating to the skin after direct contact of only a few minutes (Dow, 1974). Acute exposure to vinylidene chloride may cause moderate eye irritation. The compound affects human corneal mucosa at concentrations of 0.1 mg/l (4 ppm) (Rylova, 1953). Based on animal experiments, chronic exposure to low levels of vinylidene chloride would be expected to result primarily in liver and kidney damage (Prendergast et al., 1967). Due to its structural similarity to vinyl chloride and to some indications of carcinogenicity in laboratory animals, the possibility that vinylidene chloride may be a human carcinogen should be investigated. Professor Cesare Maltoni, who in 1972 linked vinyl chloride with

angiosarcoma, is suspicious of all compounds with a carbon-chloride link (Ricci, 1974). Despite these suspicions, there has so far been no evidence of a relationship between vinylidene chloride and human cancer.

Forty-six to fifty employees who worked with vinylidene chloride at a BASF-Wyandotte Corporation plant in South Kerny, New Jersey, were given complete physical examinations (Chemical Marketing Reporter, 1976). Results of preliminary tests indicated the desirability of administering liver scans and measuring liver enzyme activity in these workers. Positive findings for liver dysfunction were obtained for twenty-seven of these workers. Dr. C. M. Leevey, a recognized authority on liver disease, confirmed the results and concluded that ten of the workers should be given liver biopsies (Chemical Marketing Reporter, 1976). These findings did not provide sufficient evidence to demonstrate that vinylidene chloride was the cause of these irregularities.

An epidemiological study was conducted on 138 Dow employees occupationally exposed to vinylidene chloride and copolymers other than vinyl chloride (Ott et al., 1976). Time-weighted average concentrations to which workers were exposed varied from less than 5 ppm to 70 ppm. No long-term mortality data or multiphasic health effects were statistically related or individually attributable to vinylidene chloride exposure (Ott et al., 1976).

According to Jaeger (1975) data obtained from industry and the National Institute of Occupational Safety and Health (NIOSH) indicate that workers at a polyvinyl chloride facility who developed angiosarcoma of the liver were exposed to both vinyl and vinylidene chloride.

A retrospective study of Dow workers revealed several statistically positive correlations between indices of hepatic injury and chronic exposure to vinyl chloride and smaller amounts (up to approximately 5 ppm) of vinylidene chloride (Kramer and Mutchler, 1972). Concentrations of vinyl chloride at these plants ranged up to 300 ppm (Kramer and Mutchler, 1972).

2. Animals

Acute Toxicity

Vinylidene chloride is toxic to laboratory animals through inhalation or oral exposure and can be fatal at sufficiently high dose levels. The acute toxicity of vinylidene chloride is similar to carbon tetrachloride and significantly greater than the acute toxicity of vinyl chloride.

Mortality in Sprague-Dawley rats exposed to vinylidene chloride was observed by Siegel et al. (1971) during both a four-hour inhalation exposure and a subsequent two-week observation period. The LC_{50}* was determined to be 6,350 ppm. Starved rats are much more sensitive to vinylidene chloride toxicity than fed rats. Jaeger et al. (1974) found that the 24-hour LC_{50} for Holtzman rats fed ad libitum was 15,000 ppm while the 24-hour LC_{50} for 18-hour (overnight) fasted rats

*LC_{50}: mean lethal concentration.

was 600 ppm. The minimum lethal concentration was 200 ppm for fasted rats and 10,000 ppm for fed rats.

The 24-hour oral LD_{50}* for vinylidene chloride in male Holtzman rats is approximately 1,500 mg per kg body weight (Jenkins et al., 1972). Adrenalectomy 10 to 14 days prior to administration of vinylidene chloride reduced the 24-hour oral LD_{50} to 84 mg/kg.

The acute toxicity of continuously inhaled vinylidene chloride was studied by Short et al. (1977) using CD-1 mice and CD rats. He found that mice were more sensitive than rats to the lethal and hepatotoxic effects of vinylidene chloride. Male mice were more sensitive than female mice to the lethal effects of vinylidene chloride since at the end of 7 days' exposure to 40 ppm, 7/10 males were dead while no females had died. The one-day LC_{50} of vinylidene chloride was 98 ppm for male mice and 105 ppm for female mice.

Chronic Inhalation Exposure

Inhalation studies are important since human exposure is expected to occur mainly through inhalation. Animal studies have shown that toxic symptoms can result from sublethal inhalation exposures to vinylidene chloride. Even when the concentrations of vapor are too low to cause an anesthetic effect, they may produce injury to liver or kidney (Dow, 1974). Rats exposed to a vapor concentration of 200 ppm for 20 exposures of six hours each developed a slight nasal irritation (Gage, 1970). Rats exposed to 500 ppm vinylidene

*LD_{50}: mean lethal dose.

chloride showed symptoms of nasal irritation and retarded weight gain. Histological examination revealed liver cell degeneration (Gage, 1970).

Rabbits, rats, dogs, monkeys, and guinea pigs (Prendergast et al., 1967) were exposed to repeated daily eight-hour exposures of 395 mg/m^3 (100 ppm) for five days per week over a period of six weeks. No animals died. The rabbits and monkeys lost weight and one rat had bloody urine. During a continuous 90-day exposure to 189 mg/m^3 (67 ppm), 7 out of 15 guinea pigs died between the fourth and ninth day of exposure and three out of nine monkeys died on days 26, 60, and 64. Dogs and monkeys lost weight. Rats gained less than controls. Gross examination revealed mottled livers. The hepatic changes in dogs, monkeys, and rats and the renal changes in rats were considered to be a direct result of vinylidene chloride exposure. Histological examination showed morphological changes in both liver and kidney of rats and livers only of dogs and monkeys. One adrenal gland from a dog contained a cortical adenoma. There were also nonspecific inflammatory changes in lungs of a majority of animals. Activity of microsomal enzymes (liver alkaline phosphatase and serum glutamic-pyruvic transaminase) were increased in rats and guinea pigs (Prendergast et al., 1967).

During a continuous 90-day exposure to 101 mg/m^3 (25 ppm), 3 out of 15 guinea pigs died between the third and sixth exposure days and two out of three monkeys on days 39 and 47. At an exposure level of 61 mg/m^3 (15 ppm), 3 out of 15 guinea pigs died on the third and fourth days of exposure. At an exposure level of 20 mg/m^3

(5 ppm), 2 out of 45 rats, 2 out of 45 guinea pigs, and 1 out of 21 monkeys died. The dogs lost weight and rats gained less than controls. Gross examination revealed mottled livers in about one-third of the animals in all species. Histological examinations revealed non specific inflammatory changes in lungs of all species and in kidneys and livers of monkeys. Elevations in liver alkaline phosphatase and serum glutamic-pyruvic transaminase activities that had been found in the 189 mg/m^3 exposure could not be demonstrated at 20 mg/m^3 (Prendergast et al., 1967).

Since 1974, the Manufacturing Chemists Association has been administering a research program conducted by the Toxicology Research Laboratory of the Dow Chemical Company in Midland, Michigan, to investigate the effects of vinylidene chloride on laboratory animals. This program includes inhalation studies on rats. Among rats exposed to 25 or 75 ppm (6 hours/day, 5 days/week) for up to 18 months, the only significant findings were minimal microscopic changes in the liver (Norris, 1977; Rampy et al., 1977).

Liver

The liver is the prime target organ of vinylidene chloride in most laboratory mammals and, apparently, in humans. Hepatotoxicity has also been observed in perfused livers.

Hepatic injury occurs quite rapidly after inhalation exposure. Hemorrhage and necrosis were observed in liver sections of fasted rats within two to four hours of exposure to 2,000 ppm vinylidene

chloride (Jaeger et al., 1974). Oral doses of 400 mg/kg body weight were hepatotoxic to rats (Jaeger and Murphy, 1973).

Morphological changes in livers of animals (dogs, monkeys, and rats) exposed to vinylidene chloride included fatty metamorphosis, focal necrosis, hemosiderin deposition, lymphocytic infiltration, bile duct proliferation, fibrosis and pseudo-lobule formation (Prendergast et al., 1967).

Jaeger et al. (1974) observed morphological changes in livers of fasted rats. They noted that these changes were distinctive and did not resemble those which occur following vinyl chloride exposure. Early changes do not involve the endoplasmic reticulum. Striking changes in the nuclear, mitochondrial, and/or plasma membrane suggest that one or all three of these organelles are primarily affected by vinylidene chloride or its toxic metabolites.

Fasted rats are more susceptible than fed rats to hepatotoxic effects of acute inhalation exposure to vinylidene chloride. Following a 4-hour exposure to 200 ppm vinylidene chloride, pathologic changes in fasted rats included midgonal necrosis, thrombus, damaged parenchyma and a few inflammatory cells. The earliest observation of such changes was two hours following exposure. These symptoms were not observed in fed rats exposed to vinylidene chloride under similar conditions (Jaeger et al., 1975).

Symptoms of liver injury caused by both oral and inhalation exposures to vinylidene chloride have been observed during a series

of toxicology tests being conducted by the Manufacturing Chemists Association (Humiston et al., 1975; Rampy et al., 1977). Results of a 90-day study incorporating vinylidene chloride in the drinking water of rats showed minimal liver injury at a vinylidene chloride concentration of 200 ppm. This concentration was equivalent to an average daily dose of 19 mg per kg body weight for males and 16 mg per kg body weight for females. The liver injury, as revealed during microscopic pathology examinations, was in the form of increased vacuolation in the cytoplasm of the hepatocytes. Inhalation exposure of rats to 25 to 75 ppm of vinylidene chloride for 30 or 90 days also produced minimal liver injury. The incidence of liver injury was dose-related and increased with duration of exposure (Norris, 1977).

The appearance of large quantities of neutral fat within the liver is an indication of hepatotoxicity. Jaeger et al. (1973) observed an increase in the total amount of hepatic fat in rats as the dosage of vinylidene chloride was increased. At 800 mg/kg body weight, the amount of lipid present was almost double that of controls.

Perfused livers of fasted rats became pale and swollen and the flow rate of perfusate through the liver decreased when the liver was exposed to vinylidene chloride at a concentration of 20,000 ppm in the gas phase (Jaeger et al., 1974).

Vinylidene chloride can disrupt normal microsomal enzyme activity. These enzyme activity alterations are the most sensitive indicators of vinylidene chloride injury to the liver. Twenty hours

after administration of single doses of 500 mg vinylidene chloride per kg body weight, male rats showed elevenfold increases in liver alkaline phosphatase activity and thirteenfold increases in liver tyrosine transaminase activity (Jenkins et al., 1972). Both of these are stress responsive enzymes (Jaeger et al., 1973). After 44 hours, plasma alanine transaminase reached a maximum activity of 19 times controls and plasma alkaline phosphatase activity showed a maximum fivefold increase. Liver glucose 6-phosphatase activity reached maximum depression (52 percent of controls) after 44 hours and then increased. These effects resemble the biochemical effects of carbon tetrachloride (Jenkins et al., 1972).

Elevated serum alanine--ketoglutarate transaminase (AKT) precedes hepatic necrosis and death. In rat liver perfusion experiments, increased AKT concentrations in perfusate correlate well with grossly observable liver injury and are often used as indicators of hepatotoxicty (Jaeger et al., 1974). Oral administration of vinylidene chloride (100 to 800 mg/kg body weight) caused dose-dependent liver injury as measured by increased serum AKT activity (Jaeger et al., 1973).

The relationship between hepatotoxicity (measured as increase in plasma transaminase levels) and dosage level for vinylidene chloride administered to Holtzman rats in single oral doses indicates a threshold effect (Anderson and Jenkins, 1977). The transition from a dose for which the effect was relatively benign to one for which the effect was extensive hepatic injury occurred abruptly over a narrow dosage

range. In immature male rats weighing between 90 and 120 gm the threshold occurred at approximately 25 mg/kg. Females were much less susceptible to vinylidene chloride hepatotoxicity than males. The threshold dose for female rats was approximately 100 mg/kg.

Glutathione, a sulfhydryl-containing tripeptide, has been postulated to play a role in detoxification of vinylidene chloride. Animals with a diminished hepatic glutathione concentration are significantly more susceptible to vinylidene chloride (Jaeger et al., 1975). In both intact liver and isolated perfused rat liver, normally insensitive livers were rendered susceptible to vinylidene chloride toxicity after glutathione depletion (Jaeger et al., 1974).

Glutathione concentrations are lowered during fasting and during the night. Animals are more susceptible to vinylidene chloride under these conditions (Jaeger et al., 1975). Vinylidene chloride was uniformly hepatotoxic and fatal to 18-hour fasted rats at concentrations which were not fatal to rats fed ad libitum. Fasted rats have a very sharp vinylidene chloride dose-response curve: 100 ppm is a no-effect concentration, while 150 ppm causes a significant elevation of serum AKT and at 200 ppm deaths are observed. In fed rats, 2,000 ppm produced some injury but no deaths (Jaeger et al., 1974).

Fed animals are more sensitive to the hepatotoxic and lethal actions of vinylidene chloride if exposures are conducted at night. At this time, hepatic glutathione concentrations are at or near their circadian* minimum (Jaeger et al., 1973 and 1974). Enhancement of

*Circadian: pertaining to rhythmic biological cycles recurring at approximately 24-hour intervals.

vinylidene chloride toxicity due to short-term fasting or the circadian periodicity of glutathione are important factors to consider in reproducibility of toxicity testing or in industrial hygiene.

Kidney

Renal damage has been observed following vinylidene chloride exposure. Inflammation of the kidney has been observed in rabbits, rats, dogs, monkeys, and guinea pigs (Prendergast et al., 1967). Sections of kidney from rats showed nuclear hypertrophy of the tubular epithelium (Prendergast et al., 1967). These renal changes were considered to be a direct result of vinylidene chloride exposure.

Lungs

Although lung damage has been observed during prolonged inhalation exposures (Prendergast et al., 1967), the lungs are not particularly sensitive to vinylidene chloride. Carlson and Fuller (1972) observed no lung damage in rats exposed to vinylidene chloride vapor for one hour.

Central Nervous System Depression

Vinylidene chloride is a central nervous system depressant (Jenkins et al., 1972). It has been shown to be narcotic to mice, rats, rabbits and guinea pigs (Rylova, 1953). High concentrations of vinylidene chloride can elicit a strong narcotic effect leading to unconsciousness (Irish, 1967).

Two to four hours after administration of a 400 mg per kg body weight oral dose of vinylidene chloride, Jaeger and Murphy (1973) observed prolonged pentobarbital-induced sleeping time. The prolonged

sleeping time was accompanied by elevated serum corticosterone levels but hepatic injury was not observed at this time. These observations imply that central nervous system effects rather than disturbances of liver metabolism may have been involved in the prolongation of sleeping time.

Mutagenicity

Vinylidene chloride has been shown to be mutagenic in microbial systems. Bartsch et al. (1975) examined the mutagenicity of vinylidene chloride in Salmonella typhimurium strains, using a tissue-mediated assay which has been found effective in detecting the mutagenicity of various carcinogens. Liver, kidney, and lung fractions from mice efficiently converted vinylidene chloride into mutagenic metabolites in vitro (Bartsch et al., 1975). Conversion was greatest in the presence of liver fractions. With liver and kidney fractions, a much lower mutagenic response was observed using rat tissues than using mouse tissues. When the concentration of vinylidene chloride in air was raised from 2 to 20 percent, an increased mutagenic effect was noted only with mouse kidney and lung fractions.

Greim et al. (1975) also found vinylidene chloride to be mutagenic in a metabolically activated in vitro system. Mutations were induced in Escherichia coli K12 incubated with liquid vinylidene chloride (2.5 mM in media), liver microsomes from phenobarbitol-pretreated mice, and a NADPH-generating system. No mutagenic activity of vinylidene

chloride was detected in the absence of microsomal activation (Greim et al., 1975).

As has been shown for vinyl chloride, these results demonstrate that the mutagenic effect of vinylidene chloride is mediated by microsomal enzymes from various organs in the presence of an NADPH-generating system and oxygen. The enzymes of the microsomal mixed-function oxidase system probably convert vinylidene chloride to alkylating intermediates.

Addition of sulfur-containing compounds to in vitro mutagenicity assays containing mouse-liver fractions reduced the mutagenic effects of vinylidene chloride (Bartsch et al., 1975). These results indicate that the mutagenic vinylidene chloride metabolites are trapped by nucleophilic sulfur groups, thus competing for binding to bacterial DNA. This observation parallels the findings of Jaeger et al. (1974) that the toxicity of vinylidene chloride in rats is correlated with hepatic glutathione concentration, and that cysteine has a protective effect.

Results of a cytogenitic study of rats exposed to 25 or 75 ppm vinylidene chloride for 6 hours per day for 5 days a week for 26 weeks revealed no chromosomal aberrations in control or treated animals (Norris, 1977).

Carcinogenicity

Most chemical carcinogens have now been found to be mutagens, when assayed in one of the mutagenicity tests that combine

microbial or mammalian cell systems as genetic targets with an *in vivo* or *in vitro* metabolic activation system (Miller and Miller, 1971). Therefore, there is a high probability that chemicals found to be mutagenic in these tests will also be carcinogenic. Although mutagenicity in microorganisms cannot be assumed to indicate carcinogenicity in humans, mutagenicity tests are currently used to select environmental chemicals for carcinogenicity tests. Since vinylidene chloride has been found to be mutagenic in *Salmonella typhimurium* and *Escherichia coli* using a tissue mediated assay (Bartsch et al., 1975; McCann et al., 1975; Greim et al., 1975) the need for investigating its carcinogenicity is indicated.

Professor P. L. Viola of Regina Elena Institute for Cancer Research in Rome had reported in 1974 that preliminary results of his studies indicated that inhaled vinylidene chloride may be carcinogenic to rats at high concentrations (Chemical and Engineering Journal, January 20, 1975). Viola's latest findings, however, indicate that tumor incidences in Wistar and Sprague-Dawley rats inhaling vinylidene chloride were similar to tumor incidences in controls (Viola and Caputo, 1977).

Dr. Cesare Maltoni of the University of Barcelona in Italy is conducting two-year inhalation studies of vinylidene chloride using rats, mice, and hamsters. He found that 20 to 25 of 120 male Swiss white mice exposed to 25 ppm of vinylidene chloride in air for one year developed kidney carcinomas (Chemical and Engineering News, 1977;

Murray, 1977; Toxic Materials News, 1977). Female mice exposed to the same concentrations did not develop kidney tumors. Neither male nor female mice exposed to 10 ppm of vinylidene chloride developed tumors. Maltoni found no evidence of tumorigenicity of vinylidene chloride in bioassays using rats or hamsters (Murray, 1977).

Dr. Perry Gerhing, Director of Toxicology Research for Dow, disputed Maltoni's conclusions on grounds that he believes mice are peculiarly susceptible to vinylidene chloride and that the mice in Maltoni's experiments developed cancer only secondarily to compound-induced kidney damage (Toxic Materials News, 1977).

Two-year ingestion and inhalation studies on rats and a three-generation ingestion study, included in the Manufacturing Chemists Association Toxicology Program being conducted at Dow, are designed to include histopathologic examinations of tissues and organs. These examinations and data evaluation are still in progress, but preliminary results based on gross tumor count indicate no evidence of treatment-related tumorigenicity (Norris, 1977 and Rampy et al., 1977).

At Midwest Research Institute, a one-year inhalation study of vinylidene chloride was conducted utilizing albino CD-1 mice and CD rats (Lee et al., 1977). Groups of 36 males and 36 females of each species were exposed to 55 ppm vinylidene chloride for 6 hours per day for 5 days a week. In the mice exposed to vinylidene chloride, hemangiosarcoma occurred in the livers of two males and one female. No hemangiosarcomas were found in livers of control mice. Hepatomas were

also observed in two male mice and one female mouse exposed to vinylidene chloride, but were not observed in control mice. Non-neoplastic lesions observed in the livers of treated mice included enlarged and basophilic hepatocytes with enlarged nuclei, mitotic figures, microfoci of mononuclear cells, focal degeneration and necrosis. One rat exposed to vinylidene chloride developed a hemangiosarcoma of a mesenteric lymph node and another exposed rat developed a subcutaneous hemangiosarcoma. Hemangiosarcomas were not observed in control rats (Lee et al., 1977). The biological significance of the mouse hepatomas is questionable since hepatomas have been reported to occur spontaneously in mice of this age. Although hemangiosarcomas are less likely to occur spontaneously in rats or mice than hepatomas, the incidence of hemangiosarcomas observed in rats and mice was too low to be considered proof of carcinogenicity.

Vinylidene chloride has been selected for bioassay by the National Cancer Institute. It is currently on test for possible carcinogenicity in rats and mice at Gulf South Research Institute.

The National Science Foundation is sponsoring a carcinogenesis study of vinylidene chloride being conducted by the New York University Medical Center Institute of Environmental Medicine. This study is using two-stage skin tests on mice to determine if vinylidene chloride can act as an initiator or promoter of carcinogenesis or if it is a complete carcinogen. Vinylidene chloride is also being tested by subcutaneous injection.

Synergistic and Antagonistic Interactions

Because vinyl chloride and vinylidene chloride monomers are structurally similar and are sometimes used simultaneously in the manufacture of copolymers, interactions between these compounds should be considered. The interaction between these compounds appears to be time dependent. Data suggest that the vinyl chloride monomer may protect against vinylidene chloride hepatotoxicity when given simultaneously, but prior administration of vinyl chloride diminished liver glutathione concentration, enhancing the degree of liver injury (Jaeger et al., 1975).

Vinyl chloride monomer is not acutely hepatotoxic in normal rats. Modest doses of vinyl chloride given simultaneously with vinylidene chloride afford significant protection against vinylidene chloride-induced hepatic injury. The combined exposure to vinyl chloride monomer (1,056 ppm) and dichloroethylene (195 ppm) in a molar ratio of 5 to 1, resulted in complete protection of fasted rats. At a lower molar ratio (3 to 1) the protection was still apparent. Only at an equimolar concentration did injury occur to two out of the five fasted, exposed rats (Jaeger et al., 1975).

Simultaneous exposure of fasted rats to a sixfold excess of vinyl chloride monomer (12,093 ppm) completely prevented elevation of serum AKT or serum sorbitol dehydrogenase (SKH) associated with approximately 2,000 ppm of dichloroethylene. This antagonism of vinyl chloride to the hepatotoxic effect of vinylidene chloride

suggests a competitive interaction between these compounds (Jaeger et al., 1975).

On the other hand, preexposure to vinyl chloride can deplete glutathione concentrations in the liver, enhancing vinylidene chloride toxicity. Jaeger et al. (1975) observed enhanced early acute hepatotoxic response to vinylidene chloride in fed rats which had been previously exposed to vinyl chloride. This synergism is consistent with a hypothesis of a common pathway involving glutathione for both vinyl chloride and vinylidene chloride toxicity or metabolism (Jaeger et al., 1975).

Pretreatment with various low molecular weight epoxides increases the hepatotoxicity of orally administered vinylidene chloride in fasted male rats (Anderson et al., 1977). Jaeger et al. (1977) found that trichloropropane epoxide enhances hepatic injury to male Holtzman rats. Anderson et al. (1977) hypothesized that epoxides increase toxicity of vinylidene chloride by interfering with the metabolism of a toxic product of the microsomal oxidation of vinylidene chloride. Jaeger et al. (1977) suggest that an epoxide intermediate may be formed upon vinylidene chloride metabolism in the liver. They believe that monochloroacetic acid may be the toxic metabolite responsible for the observed toxicity.

Administration of the hepatic microsomal enzyme-inducing agents phenobarbital and 3-methylcholanthrene to rats increased the lethality of vinylidene chloride inhalation (Carlson and Fuller, 1972). Rats

were able to tolerate very high concentrations of vinylidene chloride for one hour. However, when animals were pretreated with either phenobarbital or 3-methylcholanthrene and exposed to vinylidene chloride for one hour, most animals died either during exposure or shortly thereafter. Surprisingly, the microsomal enzyme inhibitors SKF 525A and Lilly 18947 also increased vinylidene chloride lethality as evidenced by shortened survival time during continuous inhalation (Carlson and Fuller, 1972).

The findings of Carlson and Fuller (1972) that microsomal enzyme inhibitors increase vinylidene chloride lethality, the findings of Jenkins et al. (1972) that microsomal enzyme inducers decrease vinylidene chloride toxicity, and the observation of Conney (1967) that female rats, which have lower microsomal enzyme activities than male rats, were more susceptible to vinylidene chloride toxicity suggest that metabolism of vinylidene chloride by microsomal enzymes results in products which are less toxic than the parent compound.

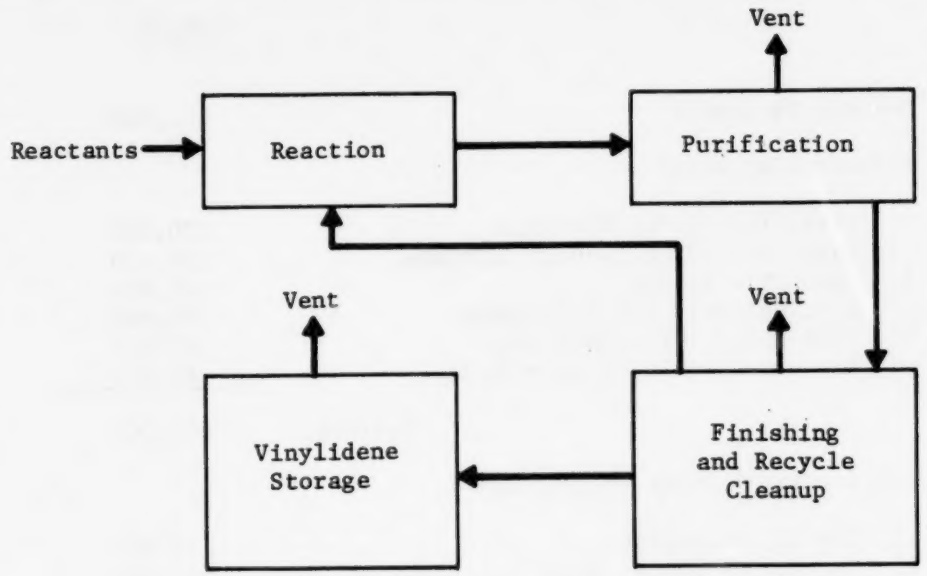
C. AMBIENT CONCENTRATIONS, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY

There are three possible sources of vinylidene chloride monomer emissions to the atmosphere during the normal production processes (see Figure 3 for the vent locations). Control devices are generally connected with the vents, but they are not 100 percent effective. The emissions from these process vents plus spills incurred during loading or unloading of transport vehicles or storage tanks would constitute most of the vinylidene chloride monomer emissions associated with its production. Vinylidene chloride is also released during the production of saran resins and saran latex as well as during 1,1,1-trichloroethane, methyl chloroform, modacrylic fiber, and vinylidene fluoride production (SRI, 1974). Releases in these polymer production facilities are summarized in Table II. Total annual releases from the polymeric uses of vinylidene chloride are approximately the same in amount as releases from the production of vinylidene chloride monomer. However, the former sources occur at many different sites (see Section D) while only three plants produce vinylidene chloride. Thus, site-specific release from polymer plants is actually far less than that for vinylidene chloride monomer production. These data are presented in Table II.

Industry initially adopted a level of maximum occupational exposure of 25 ppm (Patty, 1967). Subsequently, Threshold Limit Values (TLVs) were recommended by the American Conference of Governmental Industrial Hygienists for airborne concentrations of hazardous chemicals. Since 1974, the TLV for vinylidene chloride has been set at 10 ppm for

FIGURE 3

VINYLIDENE CHLORIDE MONOMER PRODUCTION



Source: J. P. Strasser, Dow Chemical, U.S.A., personal communication, October 21, 1975.

TABLE II

ESTIMATED ANNUAL EMISSIONS OF VINYLIDENE CHLORIDE IN THE U.S.A.

		LBS/YR	
A.	Monomer Synthesis	611,000	
B.	Polymer Synthesis*		
	1. Latex for Burner Coatings	120,000	
	2. Latex for Miscellaneous Coatings	150,000	
	3. Synthetic Fibers	160,000	
	4. Coating Resin for Cellophane	182,000	
	5. Extrusion Resin (Emulsion)	27,000	
	6. Extrusion Resin (Suspension)	40,000	
	Subtotal	679,000	
C.	Fabrication Polymer Processing*		
	1. Coating Cellophane	1,600	
	2. Coating Plastics, Paper and Glassine	16,400	
	3. Extrusion	400	
	4. Miscellaneous Coating	12,000	
	Subtotal	30,400	
	TOTAL	1,320,000	lbs/yr

Sources: J. P. Strasser, Dow Chemical, U.S.A., personal communication, December, 1975.
A. D. Little, personal communication, November 18, 1975.

*Emissions reflecting no use of control technology.

an 8-hour working exposure (American Conference of Governmental Industrial Hygienists, 1971). Sweden sets its exposure levels at a recommended 1 ppm exposure with a maximum of 5 ppm weighted average for 15-minute periods (Cook, 1974). Dow Chemical Company has stated that the average exposure of a production facility employee to vinylidene chloride monomer rarely, if ever, reaches the TLV of 10 ppm (Strasser, 1975). During those operations where higher exposure is possible, appropriate protective gear is worn. Vinylidene chloride at levels of 2 ppm has been reported in submarine and spacecraft atmospheres (Altman and Dittmer, 1968).

Determination of the risk to the general population is difficult. Ambient levels of vinylidene chloride may be estimated in the vicinity of a theoretical plant releasing vinylidene chloride through ventilation and exhaust of internal air. Atmospheric dispersion from this plant would distribute the gaseous emissions downwind, as affected by the turbulent diffusion of the air. Hypothetical plant conditions can be used as inputs to a Gaussian plume equation* from Turner's

*Ground level downwind concentrations resulting from a point source, denoted by $\chi(x,0,0;H)$ are predicted by the following equation:

$$\chi(x,0,0;H) = \frac{Q}{\pi\sigma_y\sigma_z u} \exp\left[-1/2\left(\frac{H}{\sigma_z}\right)^2\right]$$

where: Q = uniform emission rate (grams/sec); U = mean wind speed affecting the plume (meters/sec); H = effective stack height, that is, sum of stack height plus plume rise (meters); σ_y = horizontal dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters); σ_z = vertical dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters); and $\chi(x,0,0;H)$ = the ground level concentration along the plume axis in gm/m^3.

Workbook of Atmospheric Dispersion Estimates (1969). The basic diffusion equation should be modified, however, due to the effect of the plant itself on the flow of air. Mechanical turbulence in the wake of a building tends to produce aerodynamic downwash, resulting in the fairly rapid diffusion of a gaseous emission down to the ground. This region of disturbed flow extends downwind a distance equal to several times the height of the building. While this situation does not result in a Gaussian distributed plume, it is proposed that a modification of the usual formula still can be employed. This is because the turbulent mixing in the wake of the building is assumed to be distributed uniformly in the vertical direction, analogous to the situation where a plume is trapped below an inversion layer. A limited vertical mixing height can be estimated using the equation:

$$\chi(x,0,0;H) = \frac{Q}{\pi \sigma_y u (0.8L)}$$

where L is the limit of the mixing depth. In the case of an isolated rectangular building it is assumed that L equals 1.5 times the building height.

In addition, there is a horizontal wind turbulence which is assumed to result in an initial horizontal plume spread equal to the width of the building normal to the wind direction. This is analogous to an area source emission where the area equals the building top. This can be modeled using a further modification of the basic Gaussian diffusion equation. A virtual point source is assumed upwind from the building at a distance where the plume spread (for the given stability conditions)

would equal the crosswind width of the building. The modified diffusion equation is therefore:

$$X(x,0,0;H) = \frac{Q}{\pi \sigma'_y u\ (1.2h)}$$

where h is the building height and σ_y is based on the downwind distance to the receptor point plus the upwind distance to the virtual point source.*

A downwind distance of 500 meters from the emission source has been chosen as the hypothetical measurement point. This value was chosen to represent a point within the perimeter of each production facility. The values obtained for χ at this point for each of the three vinylidene chloride production facilities are given in Table III. The following equation is used:

$$\chi(mg/m^3) = 1.04852 \times 10^{-6} . \gamma\ (lbs/yr)$$

where γ is the total annual emissions of vinylidene chloride.

For the purpose of the following calculations, it is assumed that the improvements predicted at the PPG plant at Lake Charles, Louisiana are installed and function as predicted in Table III. An individual standing on the ground 500 meters downwind would receive a peak 10-minute exposure ranging from 0.22 mg/m^3 at Dow, Freeport, to 0.13 at PPG, Lake Charles to 0.11 mg/m^3 at Dow, Plaquemine. The corresponding 24-hour peak concentrations at the same three locations

*Additional assumptions: (1) vinylidene chloride monomer is non-reactive; (2) atmospheric stability class is neutral; (3) wind speed is 6 meters/sec; (4) plants are in constant operation except for 1-2 weeks/year for maintenance; and (5) the building height is 50 feet (15.25m) and width is 100 feet (30.48m).

TABLE III

EMISSIONS OF VINYLIDENE CHLORIDE

Producer	Location	Total Annual Emissions (lbs/yr)	Ground Level Concentration Along Plume Axis ($\chi mg/m^3$)	Emission in lbs/100 lbs VDC Monomer
PPG	Lake Charles, LA	175,000	0.183	0.10
Dow Chemical	Freeport, TX	289,000	0.303	0.51 - 0.48
	Plaquemine, LA	146,000	0.153	
	TOTAL	610,000		

Source: A. D. Little, personal communication, November 18, 1975.

would be 0.13 mg/m^3, 0.08 mg/m^3, and 0.065 mg/m^3. The maximum time-weighted average exposure during an eight-hour work day determined by the American Conference of Governmental Industrial Hygienists (1971) is 40 mg/m^3. When the reactivity of vinylidene chloride monomer is also taken into account (i.e., that it remains in its original form in the atmosphere for only a short time prior to oxidation), the potential hazard from emissions of monomer production is reduced. Except during adverse atmospheric conditions, the exposure levels of the nearby resident population are probably not significant.

Vinylidene chloride is not generally measured in the ambient atmosphere but several methods of measurement are available. Samples may be collected by absorption on silica gel, condensation in a cold trap, or as grab samples in vacuum bottles. These samples may then be analyzed by infrared spectrometry or mass spectrometry (Irish, 1967). Development of gas-liquid chromatography with a flame ionization detector has made it possible to rapidly detect vinylidene chloride monomer at the parts per trillion level in air, water, sediment, and blood (Dietz and Traud, 1973; Premel-Cabic et al., 1974). Once the samples are obtained, care must be taken to avoid oxidation and peroxide formation.

Air monitoring for vinylidene chloride was conducted in rural Pullman, Washington, from December 1974 to February 1975. The total concentrations of all isomers of dichloroethylene were measured at less than 5 parts per trillion (Grimsrud and Rasmussen, 1975). It is unlikely that vinylidene chloride would have been detected, however,

that far away from any industrial source since it is rapidly destroyed in the atmosphere (Dilling et al., 1976).

During 1976 and 1977, in connection with an EPA Ambient Air Sampling Program of the Research Triangle Institute, samples were taken at a number of locations over 6 states and analyzed by gas chromotography and mass spectrometry to determine ambient concentrations of specific pollutants. Measureable quantities of vinylidene chloride were detected at one of the testing sites in Freeport, Texas, and 4 of the 11 sites near Placequemine, Louisiana. Though the measurements were made over several hours, the concentration of vinylidene chloride represented an average concentration due to the breakthrough volume of the 10x cartridges used for the measurements. The values obtained ranged from 0.010 mg/m^3 to 0.0004 mg/m^3, all well within allowable limits (Dr. Eugene Sawicki, personal communication, RTI Report 1, and RTI Report 8, both February 1977).

Due to its volatility, vinylidene chloride is generally considered to be primarily an air problem. However, unspecified isomers of dichloroethylene were found in Mississippi River water by high resolution gas chromotography following polyphenyl ether absorption before and after treatment (Dowty et al., 1975). Widespread use of vinylidene chloride as a copolymer in food wraps could potentially result in the migration of minute quantities of the VDC into the food. Minimal information is available on this subject (Lehman, 1951). Unreacted monomer levels of 10 ppm have been measured in the polymer following production and may be released to the environment (Murray, 1977).

D. SOURCES

Vinylidene chloride was first reported by Reynault in 1838. He described it as a "strange new fluid." The compound, according to Reynault, had a slight odor of garlic and became cloudy on standing (Shelton et al., 1971). It was regarded as a laboratory curiosity until the late 1920s.

In 1922, B. T. Brooks announced that halogenated ethylenes other than vinyl chloride and vinyl bromide showed tendencies toward polymerization (Wessling and Edwards, 1967). This led to a stimulated interest in vinylidene chloride and the development by Ralph Wiley and coworkers at the Dow Chemical Company from 1932 to 1939 of the copolymerization and plasticization techniques. The commercialization of polymers containing vinylidene chloride under the trade name Saran began in 1940 (Dow, 1964). The constant and increasing use of saran is a result of its unique properties of resistance to chemical and physical degradation, low water absorption, and transparency (Dow, 1974).

The current domestic production level of vinylidene chloride is around 260 million pounds per year (Little, 1975). It is difficult to validate this figure. The industry does report production figures to the U.S. International Trade Commission but the figures are not publicly releasable when there are not more than three producers. There are only two producers of vinylidene chloride in the United States, Dow and PPG.

According to SRI (Brown et al., 1975), 90 percent of the vinylidene chloride produced in the United States was domestically consumed, and the remaining 10 percent exported.

Demand for saran copolymers has increased steadily since their development. Total annual world production of vinylidene chloride in 1967 was 100 to 150 million pounds. This annual production figure has approximately doubled in the last 10 years and there is a predicted growth rate of 5 to 10 percent per year for the next 5 years (Shelton et al., 1971).

Two companies produce vinylidene chloride at three geographic locations in the United States situated along the Gulf Coast (SRI, 1974). Dow Chemical Company, the major producer, has two facilities. Dow has been marketing vinylidene chloride monomer since 1939. PPG has only been marketing vinylidene chloride monomer for approximately seven years (Dehn, 1975). Figure 4 and Table IV provide locations of production sites and estimates of their corresponding production levels. PPG uses 85 percent of its vinylidene chloride monomer for production of 1,1,1-trichloroethane and sells the remainder to industry. They are not involved directly in the production of polymers or copolymers. Dow Chemical Company uses about 80 percent of its production of vinylidene monomer internally to produce saran polymers and markets the remaining monomer (Strasser, 1975).

There are also several foreign producers of vinylidene chloride. In Japan, vinylidene chloride is produced by Kureha Chemical Industries

FIGURE 4

GEOGRAPHIC DISTRIBUTION OF VINYLIDENE CHLORIDE PRODUCTION FACILITIES

TABLE IV

U.S. VINYLIDENE CHLORIDE MANUFACTURERS (1974)

Producer	Location	Estimated Production* (million lbs)
Dow Chemical, U.S.A.	Freeport, Texas Plaquemine, Louisiana	90-95
PPG Industries, Inc.	Lake Charles, Louisiana	170-175
	TOTAL PRODUCTION	260-270

Source: Arthur D. Little, Inc., November 18, 1975.

*SRI estimated 1971 total VDC production at 300 million pounds, 1972 at 330 million pounds and 1974 at 350 million pounds.

and Asahi-Dow Chemical Company for the production of saran-type resins. In Germany, Badischi-Anilin Soda-Fabrik (BASF) A. G. and Chemische Werke-Huels; and in England, Imperial Chemical Industries produce vinylidene chloride (Shelton et al., 1971; Wessling and Edwards, 1971).

A number of companies, among them BASF-Wyandotte Corp., B. F. Goodrich, Dow, duPont, W. R. Grace and Co., Morton Chemicals, and A. E. Stanley use vinylidene chloride monomer to produce polyvinylidene chloride (PVDC) latex resins. In 1973, 21.26 million pounds of PVDC latex resins were produced (U.S. International Trade Commission, 1973).

The earliest and still most widely used commercial process for the production of vinylidene chloride is the treatment of 1,1,2-trichloroethane with sodium hydroxide:

$$CH_2ClCHCl_2 + NaOH \longrightarrow CH_2 = CCl_2 + NaCl + H_2O$$

1,1,2-trichloroethane can be obtained in either of two ways: chlorination of ethylene dichloride or addition of chlorine to vinyl chloride (Shelton et al., 1971). Industrially, however, large quantities of 1,1,2-trichloroethane are generally available as a by-product from the preparation of other chlorinated hydrocarbons such as ethylene dichloride. Calcium hydroxide may be substituted for the sodium hydroxide, although the caustic is better (Shelton et al., 1971), or 1,1,1-trichloroethane for 1,1,2-trichloroethane (Wessling and Edwards, 1971).

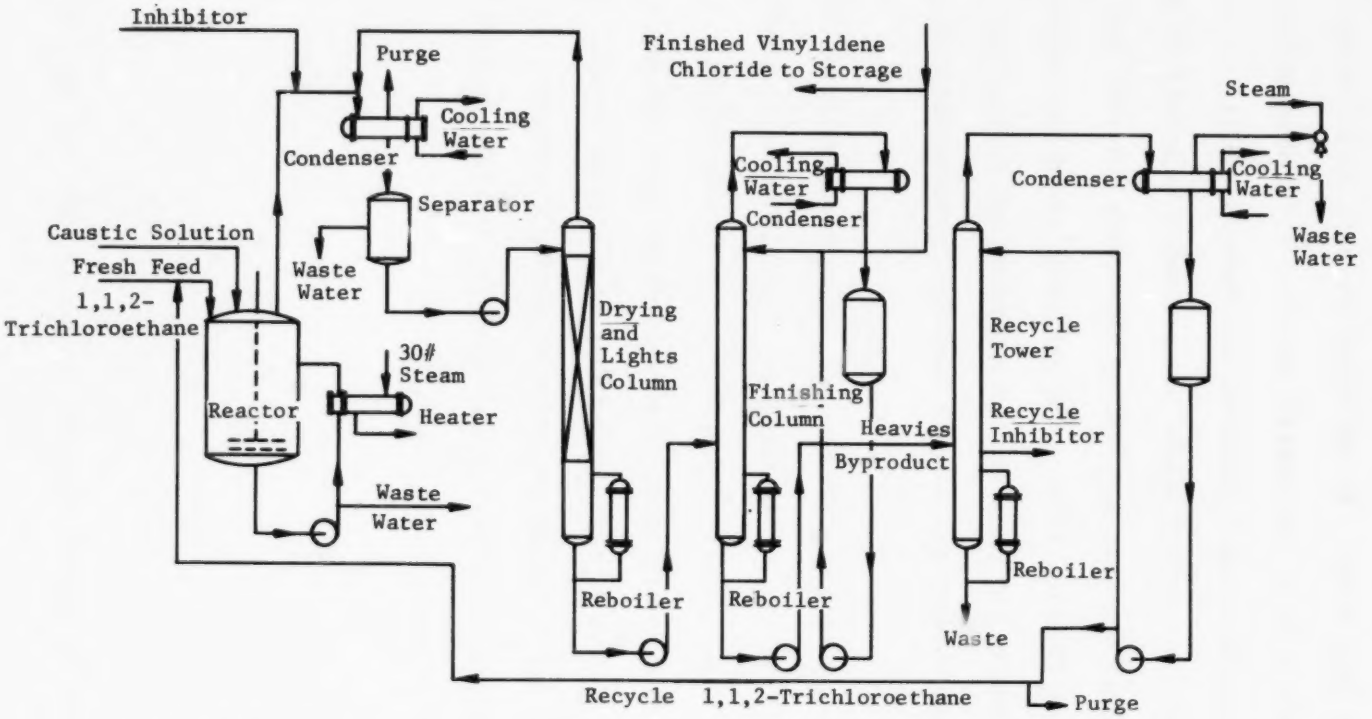
The reaction scheme is shown in Figure 5. All equipment is fabricated from stainless steel except reboiler tubes which are nonferrous. A 10 to 20 percent by weight aqueous solution of sodium hydroxide (or a calcium hydroxide slurry) and liquid 1,1,2-trichloroethane are charged simultaneously into a heated, agitated reactor where a temperature of 60° to 100°C and 15 to 30 psi of pressure are maintained (Shelton et al., 1971). As the reaction proceeds, vinylidene chloride is distilled from the batch to prevent further cracking to acetylenic compounds. An MEHQ inhibitor is generally also added at this point. Current practices favor continuous rather than batch production.

The condenser is then purged to prevent oxygen buildup and resulting peroxide formation. Water is removed after separation. The wet, inhibited vinylidene chloride is dried by azeotropic distillation and then fed to the finishing column where, under a pressure of 10 to 20 psi, the finished vinylidene chloride is withdrawn (Shelton et al., 1971). More inhibitor is added prior to storage.

The initial inhibitor, heavies and unconverted 1,1,2-trichloroethane are removed from the bottom of the finishing column and processed through the recycling tower. During recycling, waste products are removed from the bottom of the tower and the phenolic inhibitor drawn off just slightly above the bottom for recycling. The 1,1,2-trichloroethane is removed from the top of the recycling tower. After each batch is completed, a small purge of this system is also required to remove impurities which would otherwise accumulate,

FIGURE 5

PRODUCTION AND PURIFICATION OF VINYLIDENE CHLORIDE



Source: L. G. Shelton, D. E. Hamilton, and R. H. Fisackerly, "Vinyl and Vinylidene Chloride" in Vinyl and Diene Monomers, E. C. Leonard, ed., Vol. XXIV, Part 3, 1971, pp. 1205-1282.

producing chlorinated acetylenes and, hence, an explosion hazard (Shelton et al., 1971). By the above process, if it is run continuously at 98° to 99°C, the overall yield of vinylidene chloride is approximately 90 percent (Wessling and Edwards, 1971).

Domestic production losses are estimated to result in the release of 3.355 million pounds per year of vinylidene chloride into the environment* (Little, 1975). The principal production losses are through vents on the purification equipment and during recycling. There is also additional monomer loss during transportation and storage.

Additional monomer may be lost during transportation and transfer to and from storage facilities. Precautions to prevent these losses are discussed briefly in Section E of this report. It is difficult to quantify these losses. However, with the use of proper handling techniques, they can be kept at a minimal level. The industries involved in vinylidene chloride monomer production are aware of the potential handling problems and are actively enploying these techniques (Strasser, 1975; Dehn, 1975).

*This figure assumed no control technology, and thus must be viewed as an upper bound.

E. CONTROL TECHNOLOGY

Vinylidene chloride is both toxic and volatile. Immediate cleanup of spills, periodic inspection of equipment, repair of damaged equipment, and rapid repair of leaks must be the rule in order to minimize atmospheric contamination and accidental skin contact with vinylidene chloride.

In cooperation with the Michigan Air Pollution Control Commission, Dow Chemical Company has instituted controls at its new vinylidene chloride copolymer fabrication facility to maintain a release rate for organic halogenated compounds below 40 pounds per day. The facility will not contain any vinyl chloride. The controls include a hydrocarbon containment system to collect and destroy vent gases and are planned to be operational in the third quarter of 1977 (Strasser, 1977). The state of Michigan based its decision to require stricter controls on licensing practices in the state of California. These states have stricter requirements than Texas and Louisiana where the other vinylidene chloride monomer production facilities are located. Dow is planning to use the 40 pound/day emissions level as a guideline for new polymer plants dealing with halogenated hydrocarbons (Strasser, 1977).

EPA, in 1976, required that vinyl chloride in air and waste water streams remain below 10 ppm. On June 2, 1977, EPA proposed amendments in the Federal Register to the vinyl chloride standard intended to "require improved effectiveness of control technology at existing plants, impose more stringent emission limits on new sources,

and prohibit an emission increase within the vicinity of an existing source due to the construction of a new source." The proposed amendments would require existing plants to reduce emissions from the present standard of 10 ppm to 5 ppm within three years. A 5 ppm emission limit would apply to all new sources at the time of start-up and to all new oxychlorination reactors. As expected, the plastics industry is strongly objecting to these proposed changes to the 1976 standard of 10 ppm. At least part of the industry planned to adopt control measures comparable to those for vinyl chloride for vinylidene chloride (Strasser, 1975; Dehn, 1977). It is not known what effect these amendments will have on industry's plans if they are adopted.

Several methods are available to control airborne emissions of vinylidene chloride during monomer production, storage and transportation.

- Proper Design of Vents and Vapor Condensing Apparatus

Vents are located on the separator, finishing columns, recycling tower and on storage containers of normal production facilities. At worst, this vented air may contain 5 percent monomer residues (Strasser, 1975). These vents are ducted to activated charcoal absorbers or strippers (industry has not yet found a way to recycle used charcoal economically so it must be disposed of as a waste product).

A danger of peroxide formation on the surface of the charcoal exists (Wheeler and Sutherland, 1975). In work areas, aspirators can

be used to draw contaminated air to a cyclone separator and a cloth filter used to remove particulates. The air is then drawn through a blower which exhausts the monomer away from the work area, preferably to an incinerator tower. Separated particulates resulting from the filters, strippers, and scrubbers must be removed regularly (biweekly) and oxidized or incinerated to reduce any potential explosion hazard from vinylidene chloride adsorbed on the fines.

- Recovery of Vinylidene Chloride

It is possible to recover vinylidene chloride from purging gas streams used in equipment cleaning by refrigerated solvent scrubbing. The choice of solvent is largely a matter of availability. Following solvent selection, the conditions for the scrubbing are determined to minimize solvent loss with the vent gas stream. Solvents successfully used are acetone, methyl ethyl ketone, ethylene dichloride, butyl acetate, and heptyl butyl ketone. The vinylidene chloride recovered is returned to the reaction system. The resulting vent gas is free of vinylidene chloride (Wheeler and Sutherland, 1975).

- Incineration of Organic Wastes

Incinerators, or flare stacks, are widely used to dispose of flammables and should be used as a final cleanup technique for even solvent-scrubbed vent gases (Wheeler and Sutherland, 1975). Use of incineration for disposal of large volumes of vinylidene chloride would require the removal and separate disposal of the resulting hydrochloric acid.

- Proper Vent Location

Within the plant, it is important to maintain constant air flow and have filtered vents at points near where spills or leaks are likely to occur; for example, the point in the production scheme where the project quality samples are taken. Since vinylidene chloride is 3.34 times heavier than air, floor vents might be used in addition to usual wall or ceiling vents.

- Proper Storage Techniques

The monomer must be kept away fron sunlight, air, water, and other polymerization initiators. It is generally stored at -10°C, in the absence of light under a nitrogen blanket at 10 psi pressure (Dow, 1964). Care must be taken to ensure that the oxygen content of the nitrogen remains less than 0.1 percent (Dow, 1974). A water spray system should be available to keep tanks cool in the event of a fire. Dikes and adequate drainage should also be available to confine and dispose of the liquid in event of tank rupture and all wiring should be grounded (Shelton et al., 1971). Vinylidene chloride must be stored in tanks lined with nickel, baked phenolic, or glass since it may be corrosive or unstable in the presence of steel. Storage tanks should be equipped with a pressure release valve, level gauge, pressure gauge, and remote shut-off valves.

Since vinylidene chloride is volatile and is stored under pressure, it is essential that storage facilities are hermetically sealed. A melt seal may be used for vinylidene chloride as well as

vinyl chloride because mechanical sealing methods tend to fail in long-term operations. An effective seal can be easily achieved in a twin-screw mechanism through the dynamic sealing generated by reverse elements built into the screw configuration (Mack, 1975). Such a screw configuration can be readily assembled on equipment that allows interchangeable and segmented screw sections. For vinylidene chloride, Dow recommends the use of steel plug-cocks lined with Teflon plastic or ball valves with seals lined with Teflon and stainless steel balls. Gasketing materials should be made of Teflon or asbestos and pressure relief devices with a soft seal should be avoided (Dow, 1974). For vinyl chloride, EPA has recommended the use of double mechanical seals on pumps, compressors, agitators, and relief valves (EPA, 1975).

During filling of a storage tank, there is also the problem of displaced inerts disposal. Two methods are available to avoid having to vent the tank to the atmosphere. One is the use of a refrigerated vent recovery system. The second involves the compression of the storage vent stream to feed it to the plant process where vinylidene chloride is recovered and the inerts, mostly nitrogen, are removed with the production waste (Bell et al., 1975).

After extended periods of storage, vinylidene chloride monomer will often pick up iron contaminants. It is generally desirable to remove these compounds immediately prior to use. Lacquer-lined mild steel stock tanks are used when traces of iron would be objectionable.

It is recommended that the period of storage not exceed six weeks and that during storage, regular tests should be made for the development of acidity (Hardie, 1964).

- Proper Handling and Transport

To prevent peroxide formation, large-scale equipment used intermittently in conjunction with vinylidene chloride handling should be left filled with water during shutdown periods. Piping used for loading and unloading is generally purged with nitrogen and then flushed with water. When tank cars or storage tanks are used regularly, they are generally kept pressurized and checked often for leaks.

Vinylidene chloride is transported by tank truck, tank car, and 55-gallon DOT No. 17C drums used for carrying flammable liquids (Dow, 1974). During transport, the potential problem areas are sampling, vehicle emissions, vehicle gauging, and loading line clearance. Oxygen content of empty transportation vehicles should be determined remotely prior to loading since no oxygen can be permitted in the sealed vehicle and the oxygen analyzer vented remotely to avoid personnel exposure. There should also be equalizing lines so the vehicle vapor content may be displaced during loading. The final problem associated with loading operations is flushing the loading hose so that the hose may be disconnected without allowing exposure or oxygen leakage. Liquid in the loading hose must be flushed with nitrogen into the transportation vehicle. The loading line is then purged to an incinerator or tall stack or steam cleaned so the

vinylidene chloride vapors are removed (Train et al., 1973; Dow, 1974). Top-loading vehicles are recommended by Dow. Ground wires should be firmly connected to the tank car or truck (Dow, 1974).

Currently, new tank car sizes are being limited to 25,500 gallons. The car must be insulated or have heavy shields to reduce the possibility of tank punctures during derailments (Wheeler and Sutherland, 1975).

It is desirable to monitor emissions of vinylidene chloride during distribution operations. A portable organic vapor analyzer with a chromatographic column attachment can be used (Wheeler and Sutherland, 1975). Each loaded tank vehicle should be thoroughly checked for leaks with a similar instrument prior to shipment.

Pipelines offer the ultimate reduction in vinylidene chloride emissions. However, pipeline systems carrying suspected carcinogens will be subject to a stringent new set of rules requiring labeling, surface marking of underground pipes, and emergency crews on standby (Wheeler and Sutherland, 1975).

- Use of Proper Cleaning Techniques

The procedure for clearing a production equipment section for replacement involves first purging of that section of the line with high pressure nitrogen. Inerts used in the purge should then be vented to a high stack or incinerator (Bell et al., 1975). Safety precautions such as protective clothing and breathing masks followed by proper cleanup procedures should be employed. In the same way,

empty tank cars and other transportation vehicles should be purged first with inert gas and then the inert gas removed by exposure to the atmosphere. Before refilling tanks, the reverse purging process must be followed to remove the air. Tanks are often left filled with 6 to 10 psi nitrogen, but must be checked regularly.

Some loss of vinylidene chloride occurs with the waste water from the production process. Waste water is discharged from the reactor, separator, and recycling tower (see Figure 5). Waste water is also generated from tank washings and cleaning up spills. Vinylidene chloride is insoluble in water and thus vaporizes quickly. If exposed to air, these discharges could produce toxic vapors and explosive peroxides. Dissolved chlorine-containing compounds are not removed during most waste-water treatment operations. Some control measures are available to minimize these vinylidene chloride emissions and also maintain them below a 10 ppm level.

- Water Purification by Activated Charcoal

Waste water can be purified by passage over an activated charcoal slurry. The need for disposal of the spent charcoal and the potential oxidation problem were discussed previously in atmospheric control. After passage over the bed of activated charcoal, the effluent may then be altered by catalytic or thermoconversion into less harmful and more easily contained substances (Bell et al., 1975).

- Water Purification by Steam Stripping

Waste water resulting from spills or leaks should be rapidly vacuumed and sent to a steam stripper for temperature distillation. From this, the monomer can be recovered by vapor condensation or compression, and recycled (Strasser, 1975).

- Use of Waste Water in Cooling System

The purified waste water can be cycled to the condensers for use as a coolant to condense vinylidene chloride from the vapor to the liquid phase (Sittig, 1974).

- Acid Neutralization

Hydrochloric acid, resulting from incineration of organic chlorides, mixed with process water will need to be neutralized or otherwise disposed of. Neutralization of this waste water can be accomplished by passage over a limestone slurry bed (Train et al., 1973).

Since EPA has set emissions standards for vinyl chloride of 10 ppm and since in at least two of the vinylidene chloride monomer production facilities vinyl chloride is present as well, industry, for the most part, is adopting similar control procedures for the two structurally similar compounds (EPA, 1976; Strasser, 1975). Vinyl chloride is more volatile than vinylidene chloride, hence measures adequate for its control are more than sufficient for vinylidene chloride. The effect on industry of the proposed 5 ppm vinyl chloride standard is not known, but it can be expected that in

those facilities where both vinyl chloride and vinylidene chloride are present, total emissions of both would be reduced. In those facilities where only vinylidene chloride is present, control technology is still needed to maintain the required workplace exposure of 10 ppm over a 40-hour work week.

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